Editorial

Addressing Cardiovascular Disease in Chronic Kidney Disease

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Cardiovascular disease is a major cause of disease burden worldwide and the problem appears to be getting bigger [1]. The spectrum is broad and ranges from heart disease, vascular disease to cerebral dysfunction, and its connection with chronic kidney disease (CKD) is manifest in the bi-directional relationship between hypertension, a major risk factor, and the CKD itself. Thus, evidence of kidney disease was found in up to 15% of newly diagnosed patients with hypertension in a study carried out around Ibadan, Nigeria. On the other hand, CKD, and even milder forms of kidney dysfunction, are important risk factors for cardiovascular disease and all-cause mortality, as borne out by the fact that pre-dialysis CKD patients are more likely to die from cardiovascular disease, than reach end stage kidney disease.

There is, arguably, growing interest in the knowledge and research of cardiovascular disease in chronic kidney disease in Nigeria. Two articles in this issue of the journal examine aspects of this subject from different perspectives, and produce interesting conclusions or platforms upon which further studies could be built. In the paper by Hussaini et al, carotid intima media thickness (CIMT) is compared in patients with CKD stages 3-5 and controls, and a correlation is found between CIMT and kidney dysfunction, but not with blood pressure [2]. In the circumstance, the conclusion reached that CIMT can be used as a measure of cardiovascular risk does not appear very strong. In the other paper by Akintomide et al which involved CKD patients with and without cognitive impairment as determined by the Montreal Cognitive Assessment (MoCA) scores, a direct measure of cognitive function, higher SBP was seemingly over-represented in those without cognitive impairment, but no relationship was found with eGFR [3].

In both of these papers, the authors suggest the use of CIMT and cognitive function assessment in the routine evaluation of CKD patients, but considerations of availability may limit the use of carotid ultrasonography. Cognitive impairment in CKD most likely results from a number of factors including vascular disease and metabolic abnormalities; the vascular disease comprises stroke, transient ischaemic attack, microbleeds and sub-clinical stroke [4]. The foregoing, the seeming conflicting statements notwithstanding, serves to direct attention to cardiovascular disease and its influence on CKD. Incidentally however, the use of ankle-brachial index, another measure of cardiovascular disease, and CIMT has come into rather frequent and perhaps regular use in the extended assessment of cardiovascular disease in hypertension, as recommended by some hypertension treatment guidelines. Local support for the validity of ABI in the assessment of CKD comes from a study in which evidence of peripheral arterial disease was found in 54.5% of patients with CKD as against 22.7% in matched controls. Furthermore in that study, SBP, lowered eGFR and microalbuminuria were found to be correlated with ABI [5]. Should these procedures become regular tools in the assessment of CKD the expected benefits may be realized, especially with regard to the outcome of treatment. This has not always been the case though as has been witnessed before; the emphasis placed on the management of mineral and bone disease in CKD has not yielded the expected falls in cardiovascular morbidity and mortality.

References

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